

# Exercise-related blood pressure response is related to autonomic modulation in young adults: A new extension study

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## Abstract.

**BACKGROUND:** The delayed blood pressure recovery (BPR) at post-exercise has been in association with a major risk of cardiovascular disease and death.

**OBJECTIVE:** The study focused on evaluating the systolic and diastolic blood pressure recovery (SBPR, DBPR) and the autonomic modulation following treadmill exercise in healthy young adults. Although considerable literature had been published about BPR and HRV, the association between BPR and ultra-short-term HRV has not yet been completely described.

**METHODS:** Fifteen subjects performed exercise with three different intensities on a treadmill, the speed was 6 km/h, 9 km/h, 12 km/h, respectively. SBP and DBP was measured per 30 s in each trial. The synchronous 5-min electrocardiogram (ECG) signals were recorded and HRV<sub>30s</sub> parameters including SDNN<sub>30s</sub>, RMSSD<sub>30s</sub>, SDNN<sub>30s</sub>/RMSSD<sub>30s</sub>, SD1<sub>30s</sub>, SD2<sub>30s</sub> and SD1<sub>30s</sub>/SD2<sub>30s</sub> were calculated every 30 s periods in 5 min ECG signals to match the corresponding BPR.

**RESULTS:** The intraclass correlation coefficient (ICC) values and the Bland-Altman plots indicated good consistency and repeatability between HRV<sub>30s</sub> and HRV<sub>5min</sub> at three post-exercise trials, with most ICC values > 0.75. Besides, SBPR and DBPR generally decreased and returned to the Rest level in 5 mins. The Spearman correlation coefficients showed strong relationships between BPR and HRV<sub>30s</sub> sympathetic-vagal balance parameters, i.e., ratio SDNN/RMSSD and ratio SD1/SD2.

**CONCLUSIONS:** These observations represented a new insight into the cardiovascular regulation at post-exercise, which could contribute to physical exercise areas in the future.

Keywords: Blood pressure recovery, autonomic modulation, heart rate variability, electrocardiogram

## 1. Introduction

Prehypertension or hypertension is a main risk factor of cardiovascular diseases, i.e., coronary artery disease, stroke and diabetes [1,2]. The exaggerated BP response to exercise was reported to have a high incidence of hypertension due to the early alternations in cardiovascular hemodynamic and endothelial dysfunction [3,4]. Thus, a practical clinical technique to evaluate cardiovascular health is the assessment of blood pressure recovery (BPR) at post-exercise. BP response to exercise is frequently used to evaluate various aspects of cardiovascular modulation [5,6].

Heart rate variability (HRV) is a common and useful tool in assessing autonomic nervous system function, which is used in many academic areas [7]. Short-term HRV is believed to be an independent

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factor of some chronic diseases [8]. It is considered that the increasing mortality of cardiovascular diseases is related to the sudden changes in autonomic regulation at post-exercise, which could be reflected by HRV parameters [9–11]. Due to the prevalence of HRV in physical exercise, more literatures aim to pay attention to the application of accurate HRV assessment in exercise. For example, it was reported that a larger conversion in sympathetic-vagal balance was resulted from higher intensity exercise in Parekh's study [12]. Besides, it was supported that HRV monitor could help to track the exercise program of training adaptation in exercise training, and set the appropriate physical training loads to improve the performance for athletes [13–15].

BPR and HRV responses to post-exercise are related to the intensity of aerobic exercise, particularly in the first minutes of recovery [12,16]. Lewis et al. [17] found that the prolonged sympathetic predominance and slow parasympathetic reactivation at post-exercise contributed to a delayed BPR. Therefore, the investigation of the impact of exercise on autonomic modulation and BPR may provide further insight into physical exercise areas.

To our knowledge, most of the literature which bases on studying BPR and HRV at post-exercise is independent. The purpose of this study was to investigate the effects of treadmill post-exercise on autonomic nervous modulation and BPR. Instead of using the standard  $HRV_{5min}$ , the assessment of autonomic regulation was performed by using  $HRV_{30s}$ , which provided a new way to assess autonomic regulation for future research. Our research mainly aimed to investigating the BPR and the physiological mechanisms which regulated and controlled the results.

## 2. Methods

### 2.1. Subjects

Fifteen healthy were recruited to take part in this study. The measurement of the average age and the body mass index (BMI) was  $22.3 \pm 1.2$  years old and  $22.5 \pm 1.5$  kg/m<sup>2</sup>, respectively. All subjects in our study were physical healthy and not taking any medication, all of them were asymptomatic for respiratory disease and nonsmokers. All subjects were not allowed to drink alcohol or caffeine 24 hours before the study, as well as any exhaustive exercise. Each subject was informed to write a consent form prior to the experiment.

### 2.2. Experimental procedure

Before ECG signal acquisition, subjects were asked to seat quietly and breathe naturally for 10 min. Then, ECG signal was collected for 5-min duration as a baseline (Rest). Subsequently, subjects were ask to take the warm-up phase for 60 s on the treadmill, which was to ensure that the sensors were firmly attached and subjects could be familiar with the procedure. Later, ECG signals were recorded for other three 5-min periods as soon as each performance was ended. The treadmill speed was adjusted to 6 km/h (Post-E1), 9 km/h (Post-E2) and 12 km/h (Post-E3), respectively. To eliminate the impacts on the repeated measurements of HRV, the initial BP and HR prior to each trial was recorded to guarantee that cardiovascular modulation had returned to Rest condition level. Participants were allowed to maintain stable to reduce the motion artifact. Each trial lasted for 3 mins with the duration of 30 mins in-between trials and were performed in a random order.

### 2.3. Blood pressure monitoring

Brachial cuff-BP was measured by a digital BP monitor device (HEM-7211, Omron Healthcare, Japan).

Subjects were required to sit quietly with the cuff wrapped around the right arm at the same level between its center and the subject's heart. Cuff-BP was measured every 30 s and 10 cuff-BPs were recorded for BPR assessment in each trial. The values that each cuff-BP minus the mean BP at Rest condition were taken as BPR.

Specifically,

$$SBPR = SBP_i - \overline{SBP}_{Rest}, \quad DBPR = DBP_i - \overline{DBP}_{Rest} \quad (1)$$

$SBP_i$  and  $DBP_i$  were cuff-BP at post-exercise,  $\overline{SBP}_{Rest}$  and  $\overline{DBP}_{Rest}$  were mean BP values at Rest condition. The sphygmomanometer was calibrated before each trial.

#### 2.4. Data acquisition and HRV measures

In this study, the Power-Lab/16sp system (Castle Hill, AD Instruments, Australia) was used to collect and amplify the ECG signal. The frequency was set at 1 kHz. Subsequently, the signals were filtered by a 45-Hz low-pass filter and a 1-Hz high-pass filter. The ectopic beats were visually identified, then, the abnormal data were replaced by linear interpolation of adjacent RR intervals. The normal cardiac intervals were automatically measured and exported for HRV analysis.

Due to the 30 s consumption for the measurement of each cuff-BP, 30 s ultra-short-term HRV analysis was used to correlate with the BPR in this study. The consecutive  $HRV_{30s}$  was recorded to assess the autonomic alteration over time. Specifically, time-domain parameters including square root of the mean squared differences of successive R-R intervals ( $RMSSD_{30s}$ ), standard deviation of normal to normal R-R intervals ( $SDNN_{30s}$ ), and ratio  $SDNN/RMSSD_{30s}$  were calculated. Besides, non-linear parameters extracted from the Poincare plot, i.e.,  $SD1_{30s}$ ,  $SD2_{30s}$  and ratio  $SD1/SD2_{30s}$ , were calculated. Frequency-domain indicators, i.e., LF and HF, were not used in the paper as an ultra-short period (< 1 min) was too short to assess HRV frequency-domain parameters.

#### 2.5. Statistical analysis

The software SPSS Statistics (version 24.0) was used for the statistical analysis. All indices were presented as mean  $\pm$  standard deviation (SD). The normal distribution of HRV parameters were evaluated by the Shapiro-Wilk Normality Test. Repeated one-way ANOVA was performed to investigate the difference of physiological parameters prior to each trial. Bland-Altman analysis was applied to evaluate the limits of agreement between  $HRV_{30s}$  and  $HRV_{5min}$ . Additionally, the intraclass correlation coefficient (ICC) with Single Measures was used to evaluate the reproducibility of physiological indicators, i.e., HR, SBP and DBP, and the consistency between  $HRV_{30s}$  and  $HRV_{5min}$ . The relationship between BPR and  $HRV_{30s}$  parameters was examined by the nonparametric Spearman correlation test. Statistically significant differences were considered with  $p$  values < 0.05 in all tests.

### 3. Results

#### 3.1. Ultra-short term $HRV_{30s}$ as a surrogate of standard $HRV_{5min}$

The results of initial BP and HR prior to each trial are summarized as mean  $\pm$  SD in Table 1. The sensibility analysis did not show differences among trials and the ICC values for the parameters showed good consistency with ICCs > 0.831.

Table 1

The variation of HR and BP in repeated measurement prior to each trial, and the intraclass correlation coefficient (ICC) in four trials

	Rest	Post-E1	Post-E2	Post-E3	<i>p</i> values	ICC
HR	77.29 ± 10.22	76.23 ± 10.56	76.20 ± 10.98	76.92 ± 9.34	0.990	<b>0.943**</b>
SBP	116.76 ± 9.21	116.07 ± 11.40	118.21 ± 9.14	117.64 ± 8.86	0.940	<b>0.831**</b>
DBP	71.54 ± 7.86	68.79 ± 8.36	69.14 ± 8.98	71.28 ± 7.18	0.731	<b>0.906**</b>

Note: *p* values was yielded by repeated one-way ANOVA. ICC was used for analyzing the repeatability of the parameters prior to each trial. Generally, ICC values larger than 0.75 were considered good. \*\**p* < 0.01.

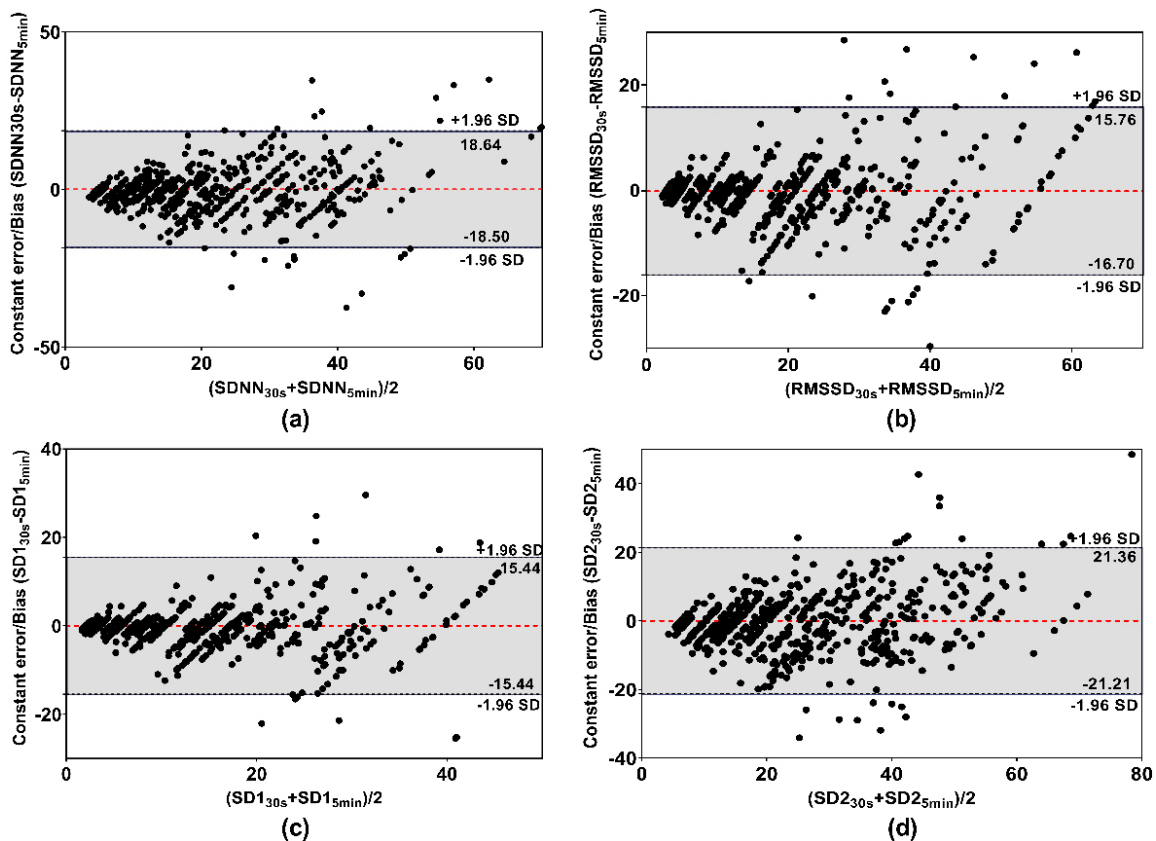


Fig. 1. The Bland-Altman analysis between HRV<sub>30s</sub> and HRV<sub>5min</sub>. (a), (b), (c) and (d) represented SDNN, RMSSD, SD1 and SD2, respectively.

Bland-Altman plots of the differences between HRV<sub>30s</sub> and HRV<sub>5min</sub> are shown in Fig. 1. The plots indicated the average difference (red line) and agreement limits (black line, ± 1.96SD) for HRV<sub>30s</sub> and HRV<sub>5min</sub>. In addition, the ICC analysis in Table 2 shows good agreement between HRV<sub>30s</sub> and HRV<sub>5min</sub> in all comparisons, with most ICC values > 0.75. These results indicated that it was acceptable for HRV<sub>30s</sub> as a surrogate of standard HRV<sub>5min</sub> in the present study.

Continuous short-term HRV<sub>30s</sub> in three post-exercise trials are presented in Figs 2 and 3 as mean ± SD. For SDNN<sub>30s</sub>, RMSSD<sub>30s</sub>, SD1<sub>30s</sub>, SD2<sub>30s</sub> and ratio SD1/SD2<sub>30s</sub>, the values were observed decreasing response to higher exercise intensity. For ratio SDNN/RMSSD<sub>30s</sub>, no significant differences were identified at three post-exercise trials.

Table 2  
The ICC results between HRV<sub>30s</sub> and HRV<sub>5min</sub>

	SDNN	RMSSD	SD1	SD2
Rest	<b>0.890**</b>	<b>0.876**</b>	<b>0.877**</b>	0.718**
Post-E1	<b>0.767**</b>	<b>0.821**</b>	<b>0.784**</b>	0.708**
Post-E2	<b>0.796**</b>	<b>0.753**</b>	<b>0.754**</b>	<b>0.756**</b>
Post-E3	<b>0.772**</b>	<b>0.837**</b>	<b>0.823**</b>	<b>0.818**</b>

Note: ICC was used for analyzing the repeatability and consistency between HRV<sub>30s</sub> and HRV<sub>5min</sub>. Generally, ICC < 0.4: poor; 0.4 ≤ ICC < 0.75: moderate; 0.75 ≤ ICC: good. \*\*p < 0.01.

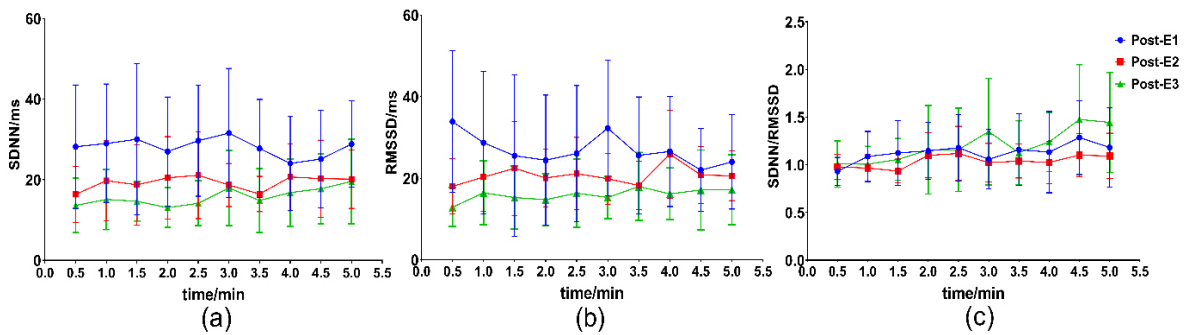


Fig. 2. The continuous time-domain HRV<sub>30s</sub> parameters at post-exercise trials. (a) is the standard deviation of all NN intervals (SDNN). (b) is the root mean square of standard deviation between adjacent NN intervals (RMSSD). (c) is the ratio SDNN/RMSSD.

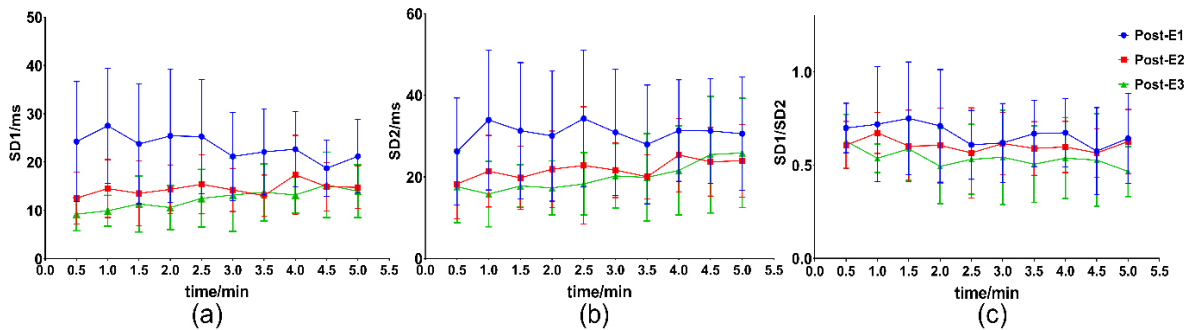


Fig. 3. The continuous non-linear HRV<sub>30s</sub> parameters at post-exercise trials. (a) is SD1, (b) is SD2, (c) is ratio SD1/SD2.

### 3.2. BPR and the relationship with HRV<sub>30s</sub>

The results of simultaneous SBPR and DBPR are illustrated in Fig. 4. As expected, SBPR and DBPR generally decreased and returned to the Rest level in nearly 4<sup>th</sup> mins at post-exercise.

There were correlations between HRV<sub>30s</sub> and BPR at post-exercise, as shown in Table 3. Spearman correlation coefficients larger than 0.6, which indicated a moderate to a strong relationship, was achieved between SBPR and ratio SDNN/RMSSD<sub>30s</sub>, DBPR and ratio SDNN/RMSSD<sub>30s</sub>, SBPR and ratio SD1/SD2<sub>30s</sub>, DBPR and ratio SD1/SD2<sub>30s</sub>. Statistical significance was determined by p values, which is also summarized in Table 3. Furthermore, these correlations are presented in detail in Figs 5 and 6 with p values and r coefficients.

Table 3  
Spearman correlation coefficients between HRV<sub>30s</sub> and blood pressure recovery (SBPR and DBPR) at post-exercise trials

	SDNN <sub>30s</sub>	RMSSD <sub>30s</sub>	SDNN/RMSSD <sub>30s</sub>	SD1 <sub>30s</sub>	SD2 <sub>30s</sub>	SD1/SD2 <sub>30s</sub>
<b>SBPR</b>						
Post-E1	0.295	<b>0.691*</b>	<b>-0.814**</b>	<b>0.626*</b>	-0.294	<b>0.607*</b>
Post-E2	-0.490	-0.229	<b>-0.714*</b>	-0.500	<b>-0.709*</b>	<b>0.618*</b>
Post-E3	<b>-0.678</b>	<b>0.733**</b>	<b>-0.831**</b>	<b>0.740</b>	<b>-0.775*</b>	<b>0.709**</b>
<b>DBPR</b>						
Post-E1	0.221	0.572	<b>-0.708*</b>	<b>0.732*</b>	-0.165	<b>0.679*</b>
Post-E2	-0.452	-0.318	<b>-0.607*</b>	-0.460	<b>-0.653*</b>	<b>0.635*</b>
Post-E3	-0.600	<b>-0.769*</b>	<b>-0.760**</b>	<b>0.905**</b>	<b>-0.710*</b>	<b>0.730**</b>

\**p* < 0.05 and \*\**p* < 0.01.

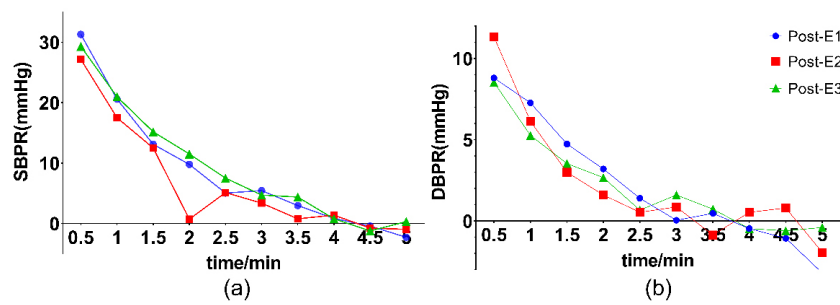


Fig. 4. The continuous blood pressure recovery at post-exercise trials. (a) is systolic BP recovery (SBPR), (b) is diastolic BP recovery (DBPR).

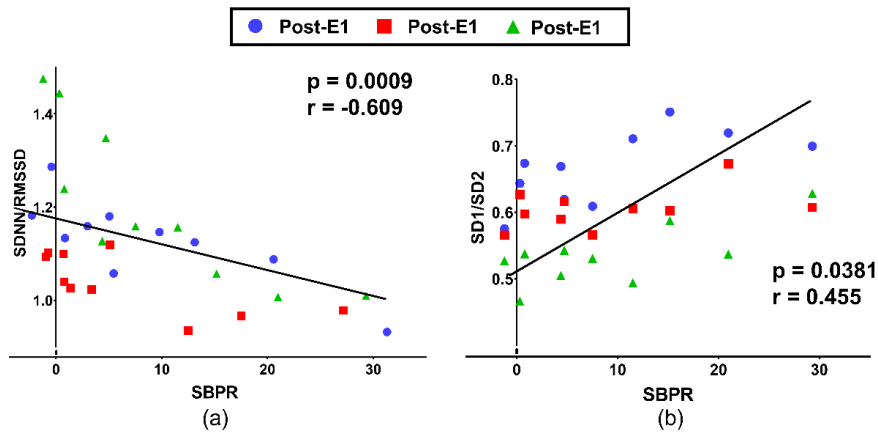


Fig. 5. The Spearman correlation between SBPR and HRV<sub>30s</sub>, i.e., SDNN/RMSSD<sub>30s</sub> and SD1/SD2<sub>30s</sub>.

#### 4. Discussion

In the present study, we investigated the effects of treadmill exercise on cardiovascular modulation and BPR during the recovery period. The continuous autonomic response was evaluated by ultra-short-term HRV<sub>30s</sub> instead of the standard HRV<sub>5min</sub>, which was a new approach to assess autonomic regulation for future research. These observations of this study were linked to the relationship between BPR and autonomic response to post-exercise.

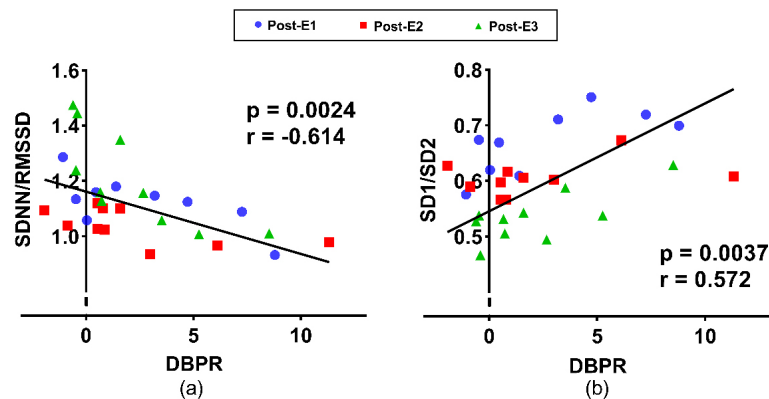


Fig. 6. The Spearman correlation between DBPR and HRV<sub>30s</sub>, i.e., SDNN/RMSSD<sub>30s</sub> and SD1/SD2<sub>30s</sub>.

Physical exercise could lead to great alteration in stroke volume and the increase of peripheral vasoconstriction. Three major mechanisms were thought to be liable for neurocardiovascular regulation during physical exercise, i.e., activation of the cerebral nervous system, the baroreceptor afferents and the activity from chemical and mechanical receptors [18–21]. The neural circuits in the medulla are activated by these mechanisms so as to regulate the balance of the sympathetic and parasympathetic tone. However, some factors can influence the physiological responses to exercise for each mechanism, i.e., exercise mode and intensity, muscle fiber type or recruited muscle mass [22]. The responses were also reflected in the autonomic alteration, as assessed via HRV parameters. Time-domain parameters including SDNN, RMSSD and ratio SDNN/RMSSD, and non-linear parameters including SD1, SD2 and ratio SD1/SD2 were utilized in the present study. SDNN and SD2 were affected by both sympathetic and parasympathetic activities [23]; RMSSD and SD1 were considered to represent vagal modulation [7]; while ratio SDNN/RMSSD and ratio SD1/SD2 could be used as an indicator of sympathovagal balance [7,24].

Instead of using the standard HRV<sub>5min</sub> to assess the autonomic alteration, ultra-short-term HRV<sub>30s</sub> was verified to be a reliable surrogate to examine the continuous change of autonomic activities at post-exercise in this study, which was an important tool in HRV monitoring applications and clinical studies [25–27]. Regarding the Rest and post-exercise trials, the concordance was observed between HRV<sub>30s</sub> and HRV<sub>5min</sub>, which confirmed that the 30 s duration could provide acceptable reliability for ultra-short time HRV analysis. The Bland-Altman analysis showed the common applicability of HRV<sub>30s</sub> by integrating all data together. The results indicated that the limits were tight for all parameters. Additionally, we intended to get the consistent results from the two different analysis methods, not just “high correlation”, so the “Absolute Agreement” type of ICC analysis was used in the study. The repeatability of HRV<sub>30s</sub> was reinforced by the ICC results with values  $> 0.75$ .

Furthermore, the decreased SDNN<sub>30s</sub>, RMSSD<sub>30s</sub>, SD1<sub>30s</sub>, SD2<sub>30s</sub> and ratio SD1/SD2<sub>30s</sub> were observed as the intensity increased, which was in line with some previous studies [28–30]. The continuous changes of HRV<sub>30s</sub> parameters mainly resulted from the enhancement of sympathetic modulation and its dominance over parasympathetic tone with the exercise intensity increasing. It had been observed by Buchheit et al. [30] and Fisher et al. [28] that the increased glycolytic metabolism was triggered by higher intensity exercise. Likewise, the activation of sympathetic activity and the withdrawal of parasympathetic tone would contribute to metabolites. However, we focused on investigating the inherent mechanisms which regulated and controlled these outcomes from the aspect of physiological significance, instead of explaining the difference of autonomic alteration of the three post-exercise trials, i.e., Post-E1, Post-E2 and Post-E3.

Physical exercise-related blood pressure response was demonstrated to have prognostic implications in clinical study. The exaggerated BP response to exercise was caused by the increased peripheral vascular resistance and impaired capacity of vasodilatation in the early period of hypertension [4,31]. BPR was extensively studied to assess the cardiovascular system. The whole exercise period was dynamic with various changes of physiological conditions. Treadmill exercise required a lot of muscle mass participation, which could produced a volume load on cardiovascular system. In general, the increase of vascular conductance and the decrease of vascular resistance were usually greater than the elevations of cardiac output after dynamic exercise, which indicated that the BPR was triggered by vasodilation [32]. On the other hand, there was an interesting phenomenon in our study: SBP had recovered to rest level at the 4<sup>th</sup> min and then maintained stability, while DBP returned to rest level at 3.5<sup>th</sup> min and decreased further. It was believed that the venous return, which stemmed from the physical vasodilation of vascular system, could led to the further decrease of DBP [33].

Besides, it had been proposed that the cerebral nervous mechanism played an important role in cardiovascular modulation, which was related to the vasodilation at post-exercise as a feed-forward system [34]. That was the reason that the correlation between HRV<sub>30s</sub> and BPR existed at post-exercise. The body stress and the metabolic status was constantly monitored by chemoreceptors and baroreceptors, which were controlled by the autonomic system. The pressure in arteries was continuously adjusted during movement as chemoreceptor and baroreceptor activities flowed into the medulla. Later, these changes were fed back to the medulla, the receptors regulated the adjusted the pressure in arteries by altering heart rate and blood pressure [35–38]. The strong correlations between ratio SDNN/RMSSD<sub>30s</sub>, ratio SD1/SD2<sub>30s</sub> and BPR indicated that BRP was regulated by the movement of sympathovagal balance. Specifically, the increased ratio SDNN/RMSSD<sub>30s</sub> and ratio SD1/SD2<sub>30s</sub> was associated with a withdrawal of sympathetic modulation and an enhancement of vagal tone. In summary, the autonomic nervous system was involved in the regulation of blood pressure and the outcomes in this study provided a new insight into the interplay between BPR and autonomic nervous alteration.

The major limitations of this study are the lack of different age ranges, i.e., younger or older participants, which indicated that the results could not be precisely extrapolated for a wider population. Additionally, only one exercise mode was performed to investigate the correlation between HRV<sub>30s</sub> and BPR. It has been demonstrated that cardiac vagal tone and vascular system had a great different response to endurance exercise versus resistance exercise [33,39]. Further research is needed to provide additional information to better understand the regulation mechanism in different exercise modes in future work.

## 5. Conclusions

In summary, exercise-related high blood pressure was a common phenomenon, which had a great relationship with autonomic modulation and vasoconstriction. The ultra-short-term HRV<sub>30s</sub> analysis was verified to be reliable to assess the continuous autonomic response to treadmill exercise in the present study. SBPR and DBPR at the post-exercise period had strong correlations with HRV<sub>30s</sub> parameters that represented the sympathovagal balance, i.e., ratio SDNN/RMSSD and ratio SD1/SD2, which was associated with the reduction in sympathetic modulation and the augmentation in parasympathetic activity.

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## Conflict of interest

All authors read and approved the final manuscript. The authors declare that they have no conflict of interest.

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